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Table 1. Independent determinants of homocysteine and transcobalamin in 55 hemodialysis patients

Dependent determinant	Independent determinant	Correlation coefficient	95% confidence interval	*P* value univariate regression	*P* value multiple regression[a]
Homocysteine					
	Creatinine	0.448	0.249, 0.685	0.0002	<0.0001
	Folate	−0.462	−0.652, −0.216	0.0005	0.0005
	Cholesterol	0.314	0.018, 0.559	0.0377	0.1268
	Triglycerides	0.279	−0.019, 0.532	0.0664	0.1417
	Albumin	0.213	−0.076, 0.469	0.1471	0.9518
	Vitamin B_{12}	−0.167	−0.418, 0.234	0.2342	
	C-reactive prot	−0.142	−0.415, 0.155	0.3496	
	Transcobalamin	0.103	−0.184, 0.373	0.4841	
	ALAT	0.132	−0.416, 0.175	0.5562	
Transcobalamin					
	ALAT	0.636	0.417, 0.784	<0.0001	<0.0001
	Creatinine	0.190	−0.099, 0.450	0.1179	0.0609
	Cholesterol	−0.225	−0.486, 0.073	0.1380	0.0655
	Albumin	−0.123	−0.391, 0.164	0.4013	
	Folate	−0.117	−0.383, 0.167	0.4197	
	Homocysteine	0.103	−0.184, 0.373	0.4841	
	C-reactive prot	−0.083	−0.367, 0.216	0.5920	
	Vitamin B_{12}	−0.076	−0.344, 0.204	0.5978	
	Triglycerides	−0.032	−0.322, 0.264	0.8375	

[a]The multiple regression analysis included only the parameters that presented a *P* value lower than 0.2 in univariate analysis.

or *MTHFR* polymorphisms, and found a weak effect of *TCN 776G* × *MTHFR 677T* allele combination on homocysteine (carriers vs. noncarriers 40.5 ± 11.3 vs. 30.9 ± 10.7 μmol/L, $P = 0.0158$). The single determinant of plasma apo-transcobalamin was alanine-aminotransferase, while creatinine was under the limit of significance (Table 1). The level was higher in ESRD, compared to 109 matched controls (719 ± 354 vs. 464 ± 160 pmol/L, $P < 0.0001$). ESRD increased plasma transcobalamin by an unknown mechanism, possibly related to glomerular filtration and tubular uptake. Under these conditions, the influence of *TCN 776C>G* on plasma transcobalamin may be limited, despite its effect on *TCN* transcription and transcobalamin intracellular concentration [3]. Therefore, in our opinion, the data of the authors are less conflicting than suggested because the weak influence of *TCN 776C>G* was initially reported in a healthy population without low serum B_{12} [1], drug use [1], and renal dysfunction [2].

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Microinflammation versus inflammation in chronic renal failure patients

To the Editor: In their study, Pupim et al [1] show that patients with end-stage renal failure (ESRF) in the highest quartile of C-reactive protein (CRP) and interleukin-6 (IL-6) before hemodialysis (HD) initiation have a significant decrease in the serum level of both markers after 12 months of HD treatment. The baseline levels for IL-6 and CRP in these subgroups were about 58 pg/mL and 62 mg/L, respectively. As the authors note, no exclusion criteria were used for enrollment in the study. These CRP and IL-6 serum levels are probably indicative of an infection or other cause of "real inflammation" (trauma, malignancy, etc.). In the referred study microinflammation investigation is warranted. It is low-grade inflammation

that is correlated to atherosclerosis or other threatening processes. Recently, a cut-off for CRP (10 mg/L) indicative for inflammation versus microinflammation was established for the general population [2]. We also found a similar cut-off for CRP and IL-6 (9.5 mg or pg/L) indicative for an inflammatory clinical event, in a group of HD patients [3]. Possibly this cut-off is higher in other HD patients, but, as the results in the majority of studies with ESRF patients show, it seems improbable to be higher than 15 mg/L for the CRP.

The study of Pupim et al is very interesting; it shows convincingly, for the first time, that HD may not be a principal cause of inflammation and oxidative stress in ESRF patients. Although possibly the results are not essentially influenced from the above-mentioned observation, accurate assessment [4] and distinction between microinflammation and inflammation is needed in all studies in this research field.

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